Post operative visual loss (POVL)

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Introduction

POVL is a rare complication after anaesthesia and surgery with increased prevalence after cardiac, spine, orthopaedic, head and neck procedures. The causes can be corneal abrasion, central retinal artery occlusion, ischemic optic neuropathy or cerebral visual loss. Anywhere in the visual pathway from cornea to occipital lobe injury can occur. The disability can range from transient blurring of vision to permanent visual loss.

Incidence ranges from 1 in 65,000 to 1 in 125000; after spinal surgery incidence is 1 in 1100. Most of these episodes do not appear to be related to direct pressure to the eye but rather to a change in the hemodynamics affecting optic nerve perfusion.

History

Corneal injuries were common during the era of ether where open drop method was the anesthetic of choice. Half opened eyes, towel covering the eyes were all contributing factors to the eye injury. Endotracheal anesthesia reduced the incidence of eye injuries. There is increase in POVL even with modern anesthetic techniques. It varies from corneal abrasions, conjunctivitis, eye lid hematoma, blurred vision persisting more than 3 hours to irreversible loss of vision.

Ischemic optic neuropathy was considered as a rare complication after spine surgery in 1950s, but in 1990 incidence has increased. Numbers of cases have increased during next decade. The reasons could be- 1) increased safety of anesthetic practice improved the visibility of complications 2) more aggressive surgical procedures 3) longer duration of surgery 4) hypotensive anesthetic techniques 5) selection of aged patients with multiple co morbidities 6) tendency to replace the blood loss with crystalloids due to fear of infections.

Pathophysiology

Causes are foreign bodies entering eyes causing corneal abrasions, dry and exposed cornea predisposing to corneal ulcers, direct long and excessive pressure on orbit or optic nerve ischemia. Blood supply to various parts of optic nerve is different. Anterior part of optic nerve (optic disc and part of nerve within sclera canal)- is supplied by short posterior ciliary arteries; additional supply from ophthalmic artery and central retinal artery is also common. Optic nerve disc is supplied by rich capillary network from circle of Zinn Halle. Posterior part of optic nerve- peripheral vascular supply only by pial branches of ophthalmic artery. Central retinal artery supplies central part of the nerve but it is much less than that to anterior part of optic nerve. Intra canalicular part of the nerve- supplied by pial branches from internal carotid and anterior cerebral arteries. Optic chiasm- supplied by internal carotid and anterior cerebral arteries Retrogeniculate optic radiation and occipital cortex- supplied by posterior cerebral and middle cerebral arteries. Retina- terminal branches of retinal artery to inner layers and choriocapillaries to outer layers. Short posterior ciliary arteries and central artery are end arteries. Each short posterior ciliary artery supplies a distinct area and watershed zones form boundaries creating precarious areas of blood supply. Types of ischemic visual loss 1) Ischemic injury to optic nerve- divided into Anterior Ischemic Optic neuropathy, Posterior Ischemic Optic Neuropathy. In AION optic disc is edematous. In PION fundal examination is normal initially but later disc pallor become apparent.
2) Cortical blindness is caused by visual loss associated with optic radiation and occipital cortex. It results from emboli, shock or cardiac arrest and is caused by damage to occipital cortex. Blindness, normal fundal examination and preservation of light reflexes are seen.

3) Central retinal artery occlusion is seen after embolic or thrombotic events and excessive extra ocular pressure. Characteristic cherry red spot is seen in retina. Central retinal vein occlusion is diagnosed with retinal haemorrhages in all four quadrants, cotton wool spots and dilated tortuous veins.

AION is due to infarction of water shed areas between zones of distribution by short posterior ciliary arteries. Asymptomatic optic disc swelling may be the early sign and may resolve spontaneously but can result in irreversible blindness. Intra operative hypotension is a rare cause. Risk factors include old age, anemia, hypertension, peripheral occlusive vascular disease, diabetes and congenital small discs. Direct orbital pressure may or may not be a risk factor. Combination of morphologically abnormal optic head with one or more vascular risk factors is sufficient to induce AION. Optical disc edema with splinter haemorrhages at the margin is seen on retinal examination. Degree of disc edema does not correlate with amount of visual loss. Disc edema resolves in 2 months and results in disc atrophy. Partial and complete recovery of vision may occur but overall prognosis is not good especially if it is a progressive form with vascular disease. Treatment includes retro bulbar steroid injection, anti platelet drugs, anticoagulation, nor epinephrine infusion, carbonic anhydrase inhibition and blood replacement.

PION presents as acute loss of vision and is due to decreased oxygen supply to posterior part of optic nerve. Blood supply to posterior part of optic nerve is from small vessels and is easily compressed by fluid accumulation in that area. Structural abnormality of optic disc is not a pre disposing factor for PION. It is not related to orbital pressure. 90% cases of POVL cause is ischemic neuropathy; 6% central artery occlusion

Risk factors
POVL is not a single entity. In few cases cause could be identified like foreign bodies causing corneal abrasions and conjunctivitis, excessive pressure on eye ball causing central retinal artery occlusion or venous thrombosis. Area of damage of optic nerve determines the prognosis.

1) Prone and lateral positions- prone position between 5 and 9 hours; increased venous pressure, increased intra ocular pressure, pressure on the eye ball, hypotension are the precipitating factors.
2) Long duration surgery
3) Pressure on the eyes
4) Intra operative hypotensive hypovolumia
5) Perioperative anemia
6) Hypotension in a hypertensive for hours leads to PION
7) Increased ocular venous pressure- central hypervolumia with hemodilution often causes facial edema
8) Trendelenberg position
9) High blood sugar decreases neuronal survival
10) Compartment syndrome within the eye has been suggested associated with facial edema and PION, increased orbital venous pressure pre disposes to PION.
11) Anaemia, blood loss and hemodilution- replacement of blood with crystalloids increases tissue edema, accentuated by trendelenberg position. This increases ocular venous pressure.excessive crystalloid infusion leads to development of compartment syndrome and PION

Treatment
Several therapies have tried but there is no definite curative therapy. Prompt evaluation, diagnosis and documentation are important.
Restoration of haemoglobin, maintenance of blood pressure, head up position, diuresis for expulsion of excess fluid give some improvement. Steroids and hyperbaric oxygen have not given consistent results. Getting an informed consent regarding the POVL and increasing the awareness among surgeons about the complication are also important.

Management
As POVL is not reversible all attempts should be made to prevent the complication. Causes are multifactorial. Trigger varies between patients. Current recommendations are:
1) History regarding previous visual problems is sought and documented.
2) Investigate and optimize diabetes and vascular disease
3) Avoid abdominal compression
4) Padding of eyes, frequent check up at intervals, document these checkups also
5) Use of Jackson frame is advisable
6) Head is positioned at or above the level of heart
7) Invasive blood pressure monitoring gives accurate changes in blood pressure
8) Blood pressure should be maintained as close to the patients’ normal level
8) Treat blood sugar above 150mg%
9) Maintenance of fluid balance
10) Minimal hemodilution
11) Timely replacement of blood
12) Reduce operating time; staging a procedure is an alternative
13) Accurate charting and recording of intra operative events
14) Post operative follow up includes attempts to assess vision and documenting; as realization of POVL may be delayed if patient has eyelid swelling, or access to the spectacles or trachea is intubated
15) Patient with facial edema should be placed in head up position, diuretics given to promote fluid excretion
16) Blood transfusion to bring the Hb back to pre op levels
17) Maintenance of euglycemia
18) Maintenance of hemodynamic stability and respiratory function
19) Ophthalmic consults as appropriate

Conclusion
POVL is multifactorial. Risk factors should be anticipated and avoided. Constant vigilance from the part of anaesthesiologist with high degree of suspicion helps to diagnose the problem. Early detection of POVL is crucial for timely consultations and proper management. Modification of perioperative management definitely reduces the incidence of POVL.

References
Sweeney PJ, Breuer AC, Selhorst JB, et al: